views & reviews

Neurologic fragments. II. Remarks on anosognosia, confabulation, memory, and other topics; and an appendix on self-observation* Not about Specilu

C. Miller Fisher, MD

Article abstract-This is an account of an informal presentation of personal views and opinions concerning behavioral phenomena-anosognosia, confabulation, memory and amnesia, perseveration and agitation, plus a few interesting brief case histories that make a neurologic point. Finally, the practice of self-observation is recommended for neurologists, and a few examples from personal experience are described.

.. NEUROLOGY 1989;39:127-132

This section is devoted mainly to personal viewpoints on anosognosia, confabulation, memory and amnesia, perseveration and agitation, but in addition includes a description of interesting cases that bear on important aspects of cerebral function. Finally, there is a brief section in which the practice of self-observation is recommended.

1. Anosognosia. Babinski¹ in his original paper used the term "anosognosia" for the patient's unawareness of her hemiplegia. It was not long before patient unawareness of blindness, right hemiplegia, paraplegia, hemiballismus, alexia, auditory agnosia, and deafness was described. Gerstmann² and Weinstein and Kahn³ extended the term "anosognosia" to denote denial of any defect or illness, and the latter authors described denial of loss of memory, incontinence, sexual impotence, recent surgical operation, and vomiting in 22 cases of brain tumor. Almost all their patients were severely deteriorated mentally. They interpreted the phenomenon as a manifestation of the patient's drive to be well, while Goldstein' spake of "the drive to selfactualization."

Unawareness accompanies so many neurologic defects that one might invoke anosognosia as a broad principle of cerebral dysfunction in humans. Anosognosia for global impairment of memory, even of moderate severity, is so common as to be the rule, particularly in Alzheimer's disease. If insight is present, it is only partial, and the deficit is greatly minimized. The denial of blindness in Anton's syndrome needs no comment. In occipital lobe blindness, a unilateral quadrantanopia is appreciated, but when both superior quadrants or both inferior quadrants are affected, the patient is unaware of blindness in the respective half-fields. The preservation of seeing in the visual field on one side in some way permits an awareness of seeing only one-half, an awareness that is lost bilaterally when vision in the second field fails. The fluent jargon aphasiac cannot comprehend why he is not understood. One aphasiac felt he was ready to return to his professional duties at a time when he referred to his glasses as his pajamas. Perseveration may not be appreciated by the patient. A brilliant scholar afflicted with aphasia from which he recovered said that "relief from the reality of the situation was built in." Dyspractics, both ideational and ideomotor, are not cognizant of their errors. The patient with auditory verbal deafness keeps on talking while the examiner is speaking and insists he can hear if the examiner speaks loudly enough. He has lost the appreciation of verbal sounds.

file: Ect: Anosugnasia

nente

Along with unawareness of his hemiplegia, the patient with a nondominant paralysis is unaware of his left visual field defect. He fails to dress the left side of his body, fits his dentures only into the right side of the mouth, and puts the right earpiece of his glasses in place, but not the left. He may read only the right 1/2 of a line of print, making no sense at all, without remarking on the incongruity. His drawings and constructions show gross abnormalities without an appropriate expression of concern. The patient with a left hemiplegia

^{*} Part I of this article appeared in the December issue.

From the Neurology Service of the Massachusetta General Hospital, Boston, MA.

Supported by The Freed Foundation, Washington, DC.

Presented in a seminar, Boston Veterans Hospital, November 13, 1987.

Received April 28, 1988. Accepted for publication in final form June 28, 1988.

Address correspondence and reprint requests to Dr. Fisher, Neurology Service, Messachusette General Hospital, Pruit Street, Boston, MA 02114.

nay lie with his eyes closed, and when asked to open them insist that they are already open. Infarction in the right anterior cerebral artery territory may be associated with a lower limb monoplegia, of which the patient is unaware. The patient with disorientation for place may claim he is in Paris, London, Timbuktu, China, or Japan on successive days, and becomes testy when challenged even slightly about such preposterous claims.

Abulia develops unbeknownst to the victim. The accompanying urinary incontinence may be in evidence in the presence of the examiner while the patient smiles. The early dement is unaware of his poor judgment, impaired concentration, impoverishment of concern, faulty dress, rudeness, and witzelsucht. It is claimed that in pain asymboly, the patient loses his appreciation of pain, although sensitive on ordinary pain testing.

There are many other examples of unawareness in which the cerebral hemispheres themselves are less clearly involved. Insight in schizophrenia is the exception. The manic depressive in the stimulated phase may buy \$100 worth of penny candy or purchase three new Cadillacs in 1 week without being aware of his state. Paranoid delusions lead to the most fantastic interpretations without insight. Grandiose delusions leave the cleverest victim bereft of insight. Some depressed patients, particularly youngsters, disclaim depression sometimes until it is very late, but this may be more a matter of terminology than of feeling. The hysteric seems to be unaware of the battle of wits going on during neurologic examination. Persons under the influence of alcohol or drugs commonly are unaware of their impairment. Dreaming proceeds without insight, and the same is often true of states of confusion. Loss of consciousness or interruption of the stream of consciousness may occur without awareness.

The Huntington's choreic may be unaware of his gyrations even though they are gross. In parkinsonism, the athetotic movements precipitated by excessive L-dopa go unnoticed by the patient. A parkinsonian tremor may be more apparent to an observer than to the patient. Likewise, the parkinsonian gait often attracts the attention of the spouse rather than the patient. Some disturbances of gait have to be brought to the attention of the victim. It may even be claimed that everyone may have a blind spot for his personal foibles. To be sure, anosognosia is broadly represented. Indeed, it may qualify as one of the general rules of carebral dysfunction.

One might ask which abnormalities arising in the cerebral hemispheres the patient is likely to be aware of. Stated simply, they seem to be motor, sensory, or emotional, and of an elementary type: dysarthria, stuttering, pure motor hemiplegia, motor aphasia, hemianopia, dysesthesias, simple hallucinations, imbalance, anxiety, fear. It is of interest that a patient with transient global amnesia senses that something is wrong, while the patient in a temporal lobe seizure usually does not.

How does anosognosia come to exist? Using the minor hemisphere as the model, it may be postulated that functions residing in the right nondominant hemispheric association areas have a neural basis and are

138 NEUROLOGY 28 January 1989

known to the rest of the nervous system via those connections operating over a lifetime, and not by visual or auditory input. The primacy of somesthetic memories may be inferred from the phenomenon of phantom limb, breast, penis, etc. When the apparatus subserving these functions is out of action, there is no way in which the rest of the nervous system can know about it. All seems to be well with the left limbs. In Wernicke's aphasia, the apparatus subserving comprehension is impaired. There is no way to analyze incoming stimuli or monitor output. In blindness, there may be no neural substrate to sense that visual stimuli are no longer being received, and in addition hippocampal memory may be severely impaired. How does one know that memory is impaired? By remembering that one has forgottenand in a memory disorder this is no longer possible. In each case, the remainder of the nervous system, the knowing part, the "I" or "me" part, the sentient part has no way of detecting and recognizing the various deficits. According to this hypothesis, anosognosia has a neural basis, and psychological factors are not operative. Awareness is primarily of elementary, motor, and sensory abnormalities, and not of more elaborate carebral dysfunction.

2. Confabulation. The discussion of confabulation has been placed immediately after anosognosia because the view to be presented here is that unawareness of the neurologic deficit, ie, anosognosia, is the circumstance under which confabulation occurs. Confabulation is best known and most developed in amnesic states where it relates to routine matters-recent activities, occupation, visitors, meals, whereabouts, and so on. Confabulation in other conditions, such as Anton's syndrome, pure verbal deafness, and nondominant parietal deficits is paid less attention. Confabulation in these states is similar in many respects, but the literature rarely provides good examples that permit comparison of the various types. Indeed, what is and what is not included under the term "confabulation" is rarely defined precisely. The cortically blind person who believes he can see, or the cortically deaf person who thinks he hears, or the patient with the left hemiplegia who thinks he moves his limbs or claims he is in a foreign land, or the aphasiac who insists that a comb is a toothbrush, is akin to the amnesiac who claims his memory is intact.

Whereas confabulation in amnesic states was formerly attributed to suggestibility, filling in, covering up, defense against the catastrophic reaction,⁴ it is now recognized as the inadvertent displacement to the present of old memories from the patient's past.⁴ In addition, obvious or inobvious perseveration and insertions and echolalia may be factors as described in item 5 in part I of this article. It is my experience that patients who are aware of their memory loss-for example in transient global amnesia, some cases of limbic encephalitis-do not confabulate, but rather that unawareness is a necessary precondition (for confabulation). Similarly, in the other states mentioned above, anosognosia is the antecedent to confabulation. The subject cannot say "I don't know" if he is unaware of his deficit. His answer is whatever comes to his mind. "I don't know" represents a relatively preserved intellect.

1000

It may be postulated therefore that confabulation is the obligatory response of the remaining functioning brain—ie, the neural patterns, the memory still existing—without recourse to psychological or voluntary factors. Innate personality traits no doubt influence the readiness of response. The term "confabulation," insofar as it connotes fabrication of ready answers and experiences without regard to truth, is misleading. A more neurologically oriented designation is in order, for example, "false recollections and delusions with anosognosia (various types)." In any case, the common connotation of confabulation should be discounted.

88

m

g

h

1

12

\$

in the second

作目

1

ĥ

3. Concerning memory and amnesia. Concepts concerning memory are commonly derived from studies of chronic stable models of amnesia, such as Korsakoff's syndrome and viral encephalitis.7 The contribution that acute processes might make are little considered. In an episode of transient global amnesia, there commonly is a transient inability to remember current items for more than 30 seconds and a transient inability to remember events of the previous 8 to 12 years, with preservation of intellectual function.8.9 The episode may last 5 to 7 hours, during which period the patient repeats a series of five to ten questions and/or comments over and over, usually in the same order, although each was answered, in turn, repeatedly. As the ability to remember immediate items returns, the ability to remember past events also returns pari passu. Episodes like this in which the memory process suddenly fails provide important clues to the normal organization of the memory process.

With transient global amnesia as the model, two inferences may be drawn: (1) When the memory apparatus or process is in abeyance, behavior becomes limited to perseverative questions and comments. During an episode, the onward flow of ideation is arrested, and the memory process is revealed as a central determinant of the ongoing ideational process. It does not determine the quality and quantity of the content of ideation, but it determines its existence and its continuity. Apparently, the memory process is subject to gradations of efficiency, failure to remember affecting all eras of past experience, the oldest being relatively spared. (2) When the memory process fails, remembering the present as well as a variable degree of the past becomes impossible (or impaired) and, of greater significance, cortical function remains relatively intact when tested by stimuli coming from the outside, ie, from the examiner. When the memory process is restored, the deficits are reversed. It may be postulated that memory is an extracortical process subject to being shut off in a uniform predictable way-a unitary nonspecific process whose normal function is to facilitate the essence of remembering—namely, the spontaneous interassociation, the interlinking of the cortical patterns or traces corresponding to life's experiences. Presumably, it is the hippocampal system that serves this function by participating nonspecifically in cortical transactions. In the operation of such a uniform system, special faculties for registration, storage, consolidation, and recall do not find a place.

The explanation for the relatively lesser vul-

nerability of older memories remains elusive. Is it possible that recent events require more hippocampal system activity to maintain the multitude of memories of the past few hours than do old memories? We can recall the details of our minute-to-minute activities of 6 to 12 hours ago; with relatively little effort as many as 200 to 400 bits of our activities can be recalled. Yet we can recall relatively little from 2 days or 1 week ago. Loss of memories predominates vastly over preservation of memories. According to such a formulation, impairment of a uniform hippocampal memory system would affect recent memories disproportionately.

C

d

d

1

D

d

d

P

p

0

i

у

u

n

0

t

1

t

0

i

İ١

r

li

V

i

i

8

d

d

V

p

4. Perseveration. Having just dealt with transient global amnesia in its amnesic aspects, it is appropriate to discuss its other prominent feature, the repetition of questions and comments during the spell. In item 7 of part I, it was mentioned that in transient global amnesia, the patient repeats the same questions and comments over and over for up to 7 hours, although at the time current memorization is almost zero. How is this particular behavior to be characterized? The nervous system is persevering inappropriately in an act, and the term "perseveration" would seem to be applicable. Sandson and Albert,¹⁰ on the basis of performance on psychological tests, recognized three types of perseveration-recurrent (aphasia), stuck-in-set (frontal lobe), and continuous (nondominant hemisphere). The repetitive performance in transient global amnesia does not readily find a place in this classification.

Were the term "perseveration" expanded to include other such instances of repetitive performance, the perseverative process would find broader representation in neurologic disease. The repetition of questions and comments that occurs in senile dements with poor memory was mentioned in item 7, part I. "Insistence." it was suggested, was a form of ideational perseveration. In ideational apraxia, perseveration may be prominent. Visual perseveration is a well-known phenomenon in occipital lobe lesions. In states of confusion, perseveration may be recognized. A patient in an agitated delirium talked rapidly and constantly, ranging widely. Her speech was recorded for several hours a day for 5 days, and analysis showed that its content was limited to three main topics or ideas, which were verbalized repeatedly. When nighttime sleep is disturbed and a dream is experienced, the same dream theme returns when sleep is resumed. In aphasia of sudden onset, the word being spoken at that moment may become "entrapped" as the only preserved utterance. In a personal case, the patient was saying the word "nothing" when she became mute as a result of cerebral embolism. Recovery was good, but 2 years later speech was sprinkled with the involuntary utterance "ting" or "ting-ting." Perseveration was semipermanent.

The obsessional activity of early Alzheimer patients may be perseverative. One spent his time copying down the serial numbers of the tires on cars parked on his street, lying underneath on the pavement using a flashlight for illumination. Another kept track of the outdoor electric meter readings of his neighbors. Still another kept detailed written lists of what he said, what was said to him, and what he wore. One item is his diary consis-

u ها 8 N E V II p 8 n р b С d t d 1 n d d p p Q i У U n 0 8 F Ft J r t Ņ r 0 i t İ١ Ð li ¥ i i s d d e a n V e

Ð

8; p

0

ted of "one shirt, Van Heusen, white, 35 long, button down."

In this wider perspective, perseveration can lay claim like anosognosia to being one of the important general principles of cerebral dysfunction.

5. Concerning restlessness and agitation. Demented patients are frequently restless, constantly moving about, pacing up and down, wandering at night, wanting to go out, and so forth. This might be viewed as abnormal, but another interpretation is that it is "normal" for human beings to be active, as is evident in children. With intellectual development the tendency to motor activity comes under control, only to reappear with intellectual decline. Affected individuals have usually been dynamic and energetic, and it is natural for them to be physically active. Many patients continue to walk several miles daily, play tennis or golf, or even skate. In brief, in dementia activity may be normal insofar as it is normal for the nervous system to be doing something, a point of view that may permit more logical management. Agitation is incompatible with abulia.

Too often agitation is regarded as a single undifferentiated state varying only in severity. Based on the observations recounted in this paper, the following classification of neurologic agitation and restlessness is offered: (1) normal vitality in dementia; (2) balking; (3) insistence; (4) anger associated with aphasia; (5) reaction to being restrained or handled; (6) calling out when isolated; (7) associated with the delusion of having to go somewhere or do something, etc, as described in item 13 of part I; and (8) fear in acute confusional state.

6. The selective localization of function. The idea that a cortical function can be highly localized is commonly met with strong rejection. In the following case, the restricted nature of the deficit is instructive. A right-handed man, age 53, suddenly developed a headache, and the cerebrospinal fluid was bloody. On admission to the hospital 1 hour after onset, neurologic examination was normal. While the examiner was writing his note in the record, the patient's wife intruded to remark that he had difficulty with names. It was explained this is not an unusual symptom. A little later, his wife again interrupted with the same information, which was not heeded. Still later, she returned to say her husband could remember her name, but not that of any other family members. This occasioned further testing, with the finding that the patient could give no single proper name, other than his wife's. He could not name his children, relatives, doctors, political figures, colleagues, churches, cars, towns, or streets. By contrast, he quickly named more than 150 pieces of furniture, clothing, and personal possessions. Comprehension was perfect, and speech production was normal in every respect. While in hospital, he kept an extensive written diary that was flawless. Reading was rapid with normal comprehension. Calculation was good. He could visualize persons he could not name. Interpretation of pictures was good, except for the naming of persons. Memory was intact. The patient's personality was not altered. There was no abulia. A cerebral angiogram was normal. The clinical picture remained the same for about 8 days, and slow improvement followed. The

130 NEUROLOGY 38 January 1949

patient returned 3 months later with signs of a left temporal lobe tumor, which proved to be a glioma. An accurate localization of the initial hemorrhage into the tumor was not possible. This remarkable example of anomia for proper names occurring in isolation indicates that a function may be selectively localized to a precise anatomic location.

7. The failure of gesture in aphasia. An extremely deaf man, age 66, with atrial fibrillation was examined neurologically following a brief transient ischemic attack. Because of the inconvenience of shouting every instruction into the patient's ear, the examiner deliberately decided to see how efficiently a complete neurologic examination could be carried out by gesture and example. The patient performed correctly and quickly beyond all expectations. The examination proceeded quickly and smoothly, and every gesture was readily comprehended. It happened that 1 week later, the patient was admitted because of the sudden onset of a severe Wernicke's aphasia, unaccompanied by limb pa-. ralysis. The patient was fully alert and on his own undertook to close a window. The rare opportunity was presented of repeating the examination of 1 week before. The patient appeared not to respond to any gesture, even the simplest, despite repetition and patience. It was a unique example of having carried out a control examination before the onset of a localized lesion.

8. Factors in pain localization. The factors that determine the localization of pain in chronic pain syndromes with a central component are largely unknown. The following case bears on this matter. A man, age 27, suffered avulaion of the left brachial plexus with a flail arm, as a result of his motorcycle striking a tree. From the beginning, there was intense pain from the shoulder to the fingertips. After 15 days, the arm pain had subsided, but a strong pins and needles sensation remained in the hand and fingers. In addition, there was an intense burning pain at the margin of the thumbnail on the side toward the index finger. It was sharply localized to a spot about 1.5 mm in diameter, exactly at the site of a stinging pain that the patient, a lifelong nail biter, regularly produced when he tore a sliver of nail from the underlying quick. The pattern of neural discharges subserving recurrent pain over a period of years must have left in its wake focal changes that predisposed to the recreation of central pain. In this regard, anginal pain can "borrow" the referred pain distribution of chronic cer-16.1 vical disk disease and histus hernia.

Impressions from self-observation. In closing, I would like to recommend for your consideration the casual avocation of engaging in observations of one's self. Although it might be viewed as an indelicate matter, I think there is a place for the scrutiny of personal experience and for self-experimentation. Physicians, particularly neurologists, are in an advantageous position to utilize the analysis of personal events for insights they might provide into the biology of the nervous system—migraine, pain, referred pain, malaise, emotion, depression, mood, worry, headache, discovery, ideas, thinking, diagnosis, and so on. There is, of course, the danger of generalizing from what may be a unique observation in a single individual, but that abould not invalidate the process.

Ĩ-

d d

-

y

1-

d

y d

y

-

8

a

Ą

». 4

r

þ

a d

£

:,

3

f

9

1. Concerning the localization of pain. It is not well appreciated that localizing an aching pain by describing where it is felt does not achieve appropriate accuracy. Especially is this true on parts of the body not ordinarily visualized, for example, the back of the head and trunk. It is essential that the site of pain be identified by the probing finger of the subject himself or the examiner and marked with a pen. Furthermore, the site should be marked at a time when pain is occurring, for after the pain has abated, it is impossible to recall its exact site. A headache felt to extend across the forehead may on careful examination prove to be mainly unilateral. To develop a special experience with pain, proper localization comes first, and its locale should be portrayed in a drawing.

2. Extinction or displacement of pain and itching. The displacement of a chronic pain syndrome by the new onset of pain elsewhere in the body is a welldocumented phenomenon.¹¹ The meaning of this datum in the biology of pain remains unclear. For example, for 15 years a middle-aged woman constantly complained of unrelieved chronic neck pain of uncertain origin. She then developed acute low back pain, requiring many different therapeutic approaches. In the first year of her new pain, neck pain was not mentioned unless inquired about.

Itch follows a similar rule, but on a moment-tomoment time scale. Itching at one site predominates over itching elsewhere, whether on the same or opposite side of the body. The organism cannot simultaneously process itching arising at more than one site. Sensory processing is tandem. In an animal, scratching with the two hind legs simultaneously would not be possible. These observations are, of course, relevant to the phenomenon variously termed "sensory extinction," "inattention," and "suppression." There are no data on whether, in right-handed individuals, itching on the right side predominates over that on the left side. Another feature of the itch sensation is that at any one instant it is highly localized, almost to a point, despite the impression that a more diffuse area of the skin is itching. The scratch reflex of a dog is accurately directed. The itch stimulus is of special interest in that like the stimulus of local pain, it arises within the akin without an external tactile component.

3. Malaise. This must be a neurologic syndrome, but its characteristics have been only vaguely delineated. In its mild form, the symptoms include tiredness, even sleepiness, an inclination to sleep longer and more deeply, not feeling well, not feeling like doing anything, decreased alertness, energy, and initiative, a lack of enthusiasm and cheerfulness, diminished appetite, and a disinclination to clear thinking. The voice may be less robust, and the body thermostat is not finely tuned. What neural system or systems are involved? Several elements of a slight depression are present, which may suggest that the toxin acts to excite the depression system, but since infection and fever are generally not precipitants of depression, it is likely that the toxin acts on more centrally located systems, as evidenced bysleepiness, chilliness, and lethargy, tilting the balance in the direction of depression.

Major malaise may be completely disabling, undermining strength, energy, initiative, appetite, alertness, and intellectual effort. It may seem trite to raise these matters for discussion, but the medical and neurologic literature is almost silent on this universal ingredient of sickness.

4. Dreaming. For some years, as convenient, I have made notes about my dreams. In every instance, the dreams made no sense. Individual parts or scenes of a dream are in themselves very well-formed, realistic images from memory, but they combine to form a ridiculous hodgepodge of loosely related or disconnected fragments. The self is always present, sometimes as a witness, most often as a participant. The emotional tone is generally low key. Sleep and dreaming are sometimes used as a model for the hallucinations and delusions of delirium and acute confusional states, in which according to theory the dream-like process is dissociated from the wake-sleep mechanism. They both reflect irrational autonomous ideational activity residing in the cortex, usually to the exclusion of external stimuli and in the absence of normal cognition and insight. In delirium, however, the emotional tone is usually intense and the content or theme more coherent than that of a dream. It is my impression that the ideation of the acute confusional state is not a good facsimile of dreaming, although the neural basis of both is related to the activity of the sleep-wake matem. This interpretation finds support in the action of barbiturates and alcohol; both are soporifics whose withdrawal may result in acute confusion.

Another feature of dreaming is that the dreamer's arms, for example, may make a quick coordinated movement, as in catching a ball, yet the position of the arms on stirring is such that they could not possibly have moved even slightly. The sleeping brain can create the vividly perceived sensation of a coordinated personal movement without motion. This experience may bear on the hallucinations of sleep-paralysis, the imagined voluntary movements of the limbs in left hemiplegia with anosognosia, and the ability of handicapped persons, for example with paraplegia or dynarthria, to perform normally in their dreams.

5. Night starts. These localized sudden muscle contractions that occur normally during the induction of sleep have been a source of several views concerning their nature.^{12,13} It is my impression that they occur in muscle groups whose relaxation at the moment of sleep will allow the part to drop under the influence of gravity or pressure, ie, the proximal limb musculature, the abdominal wall, and so forth. The contraction or jerk is the reflex response to a minimal downward motion. That it is closely linked to the onset of sleep is attested to by the fact that the thought in mind at the moment cannot be retrieved.

6. Counting sheep. This advice to the insomniac is not sound. On the verge of actually falling asleep, hypnagogic visual hallucinations may become unusually vivid, and if at that moment sheep were visualized, the idea that counting sheep to promote sleep would seem reasonable. The error lies in the fact that the threshold of sleep may produce the appearance of sheep, but imagining sheep will not educe the threshold of sleep.

7. Slips of the tongue. Ordinary verbal alips of the tongue involve words of the same category, somewhat similar to the rules that hold for some dysphasic errors.¹⁴ For example: cold for hot, week for year, higher for lower, chair for table, breakfast for supper, father for son, floor for ceiing, etc. The word may be a noun, adjective, adverb. The substituted word is well formed. Ideation seems to be intact.

Although trying to translate these events into cerebro-electrical terms is probably an exercise in futility, one might suggest that words with close natural association (hot-cold, week-year) are located in close proximity, and the "wrong" selection is the result of the dysfunction of a more general operative since so many categories can be similarly affected. No other accompanying defect that might shed light is obvious. A common trait that might be a candidate is difficulty in recalling proper names, in which no substitute name emerges but only a "blank," that actually becomes filled with the correct name after a short delay.

8. The replay phenomenon. It is not an uncommon experience that a few bars of a familiar tune will suddenly "out of the blue" intrude into consciousness 24 to 72 hours after last being heard. The music re-heard is a well-performed exact replica. The intrusion occurs while the mind is busy and is not precipitated by identifiable factors. Auditory re-creation after longer intervals has not been recognized. A somewhat analogous process occurs in the visual sphere in the form of prominent visualizations of the daytime returning at night on closing the eyes preparatory to but well before the verge of sleep. Stretches of a paved highway, rural scenes, or sports action may appear rather vividly up to 8 hours after the day's events. Occasionally, a vivid scene from 2 or 3 days before will appear. These respectments are uninvited and are not part of reverie.

Patterns of activity are capable of reappearing on the neural network after a delay and without recognized precipitating factors. This phenomenon we would term "replay." One of the theories of the indelibility of memories holds that patterns of neural activity once initiated play constantly forevermore.16 The above observation would support such a hypothesis, although serious objections to the theory can be raised. A patient of Leigh et al¹⁶ with oculogyric crises, in the period of thought disorder preceding the attack, experienced visual hallucinations "as if a short video clip were being played over and over again." Visual hallucinations of that type are most unusual and probably conform more to the phenomenon of perseveration than to replay as described berein, but at present definitions are not precise. 11

It often happens that a person's name cannot be recalled, yet minutes or hours later comes "out of the blue" when the mind is occupied with another matter. Apparently, the "search" continues or recurs, suggesting that replay of a pattern occurs until the query is THE A LAR AND . 11 ... answered.

This account of personal experience may seem paltry, and for this I apologize. Its purpose is to remind neurologists that unless they are prepared while still young to keep a diary of their experiences and illnesses, a lifetime of potentially valuable, intimate information collected by a knowledgeable observer will be lost. arte S. Gand & West

References

4.14

- 1. Babinski J. Contribution à l'étude des troubles mentaux dans l'hémiplégie organization cébérale (anosognosis). Rev Neurol (Paris) 1914:22:845-848.
- 2. Gerstmann J. Problem of imperception of disease and of impaired body territories with arganic lesions. Arch Neurol Psychiat 1942;48:890-915.
- 3. Weinstein EA, Kahn RL. The syndrome of anosognosis. Arch Neurol Psychiat 1200;84:772-791.
- 4. Goldstein K. Cited in Critchley M. The paristal labes. London: Edward Arnold, 1963:250.
- 5. Zangwill OL. Discrimination for age. J Mant Sci 1953;99:698-701.
- 6. Talland GA. Deranged memory. New York: Academic Press, 1966.
- 7. Parkin AJ. Amnesic syndrome: a lesion-specific disorder? Cortex 1984;20:479-508.
- Fisher CM, Adams RD. Transient global amnesia. Acta Neurol Scand [Suppl 9] 1964;40:1-83.
- 9. Fisher CM. Transient global amnesia: precipitating activities and other observations. Arch Neurol 1982;39:605-608.
- 10. Sandson J, Albart ML. Perseveration in behavioral neurology. Neurology 1907:37:1736-1741. Neurology 1987;37:1736-1741.
- 11. Editorial. Lancat 1976;1:945-946.
- 12. Symonds CF. Nocturnal myoclonus. J Neurol Neurosurg Paychistry 1953;16:138-171.
- Budden bodily jerks on falling asleep. Brain 13. Oswald 1959:8: 03.
- 14. Geschwood N. Varieties of naming errors. Cortex 1967;3:97-112.
- 15. Russell WR. Brain, memory, learning. A neurologist's view. Onford, UK: Clarendon Press, 1959.
- 16. Leigh RJ, Foley JM, Remler BF, Civil RH. Oculogyric crisis: a syndrome of thought disorder and coular deviation. Ann Neurol 1987;22:18-17. tin 's 111.1

4.11

it.

. .

2.41

. . .

41. 11